and his last kidney wound became a foul, sloughing cesspool, while his temperature was continually mounting. A blood transfusion changed this individual from one absolutely lost to a man of hope; his temperature dropped, the wound in his side gradually cleared, healthy granulations sprang up, and he left the hospital in good shape not many weeks later. And I maintain that is a triumph of the most fundamental character.

In conclusion, I would urge that henceforth transfusion be not regarded entirely as a measure of last resort—food for the reporters by spectacular stories of the dead brought back to life. A more thoughtful attitude concerning blood loss from any cause is greatly to be desired, and the phenomena accompanying bleedings of all magnitudes should be carefully noted and studied, with the view to improving the general knowledge of such matters. Furthermore, a partial revision of the existing ideas of combating hemorrhage and the condition commonly known as shock may possibly be of material advantage in view of the fact that transfusion may be done so readily at the present time. But above all, surgeons and physicians should learn to recognize the limits of bleeding and to act promptly. In the acute hemorrhages, according to my experience, the safest guide is the blood-pressure; in the chronic bleedings and the anemias it is the hemoglobin; but one must never lose sight of the fact that at times all signs fail, and there remains naught for guidance but experience and judgment. Under these circumstances my advice is, "When in doubt, transfuse!"

THE MAIN FACTORS AFFECTING THE INTENSITY OF SOUNDS AS THEY PASS FROM THE INTERIOR OF THE LUNGS TO THE PERIPHERY OF THE CHEST.

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The intensity of sound as it passes between two places may vary in one of three ways: It may become intensified, it may remain constant, or it may be diminished. In these general features sound presents certain resemblances to light. Factors operating to increase sounds between the interior of the bronchi and the periphery of the chest are little understood and are probably of very minor importance, as far as the mere intensity of audible sounds is concerned. Factors concerned in maintaining the original intensity of sounds are found particularly within the bronchi, which act much as speaking tubes in preventing, through limitation of diffusion, the weakening of the sounds coming from the upper respiratory tract, while at the same time the bronchi carry the sounds deeply within the lungs, thus preserving a good deal of their original intensity. The bronchi, however, provided they are patulous, and not too far retracted toward the root of the lung, exert their influences on the sounds within them under both normal and pathological conditions, so that to a large extent our interest centres on the sounds *after they have escaped from within the bronchi.* It is not, however, with an increase in sounds, or their retention, that we are chiefly concerned, but with those factors tending to effect a diminution in the intensity of sounds in the chest.

THE DIMINUTION OF SOUNDS. The diminution of sounds in different clinical conditions as they pass from the interior of the bronchi to the periphery of the chest, depends chiefly on one or both of two factors: *the diffusion of sounds* and *the reflection of sounds.*

The Diffusion of Sounds. By diffusion is meant a spreading out of the sound, a dilution of the sound energy. If a sound originates at a point, and, owing to the non-existence of any obstructions, spreads out equally in all directions, the intensity of the sound will be the same on all points of the surface of a sphere whose centre is at the point of origin of the sound. At the surface of a second sphere, double the area of the first, and concentric to the same point of sound origin, the vibration energy, per unit of area, and hence the intensity, is halved. This is a deduction from the so-called inverse square law of acoustics, which tells us that, in a medium in which the absorption of energy is small enough to be neglected, and which is extended enough (infinite) so that the complications introduced by boundaries may be disregarded, the intensity of a sound varies inversely as the square of its distance from the source. The importance of the factor of diffusion of sound in diminishing the intensity of sounds as they approach the surface of the chest has, for the most part, been understood in only a very general way, and the great relative importance of this factor in certain clinical conditions has been largely overlooked. Thus, the main factor in determining the diminution of vocal resonance in pleural effusion as contrasted with the normal lung, is diffusion.

The Reflection of Sounds. The reflection of sounds is a form of sound obstruction, and is as different from the diffusion of sound as the reflection of light is from the diffusion of light. Of course we are now speaking of reflection as it interferes with the transmission of sounds on their way to the hearer who is situated distal to the point of reflection, for reflection may intensify sounds by actually focussing them to a smaller area. In the chest we look for factors reducing sounds (exclusive of diffusion) either within the different individual media encountered, or at the junction of different media, that is, where different media occur in combination.

THE DIFFERENT INDIVIDUAL MEDIA CONCERNED. All the individual media with which we are concerned may be divided into three groups: (1) the solids, or, better, the gels, (2) the fluids, and (3) the gases. It is very easy to show that *each of these individual media by itself offers but little obstruction to the passage of sound through it, and that whatever proportion of sound is lost in its passage through these individual media is due to diffusion.* In the de-intesification of sounds on their way to the periphery of the chest, no differences in the conductive properties of any of these media, solids (or gels), fluids and gases, have been determined by the stethoscope, and therefore are not of present concern to us.

Different Media in Combination. Apart from diffusion the chief factor in diminishing sounds passing through the chest is the reflection of sounds occurring at the junction of certain of the above mentioned media. *Comparatively little sound is lost in its passage between fluid and tissue (or solid), because their densities are not greatly different and they are both highly elastic. But between air and tissue or air and fluid, provided the body of tissue or fluid is not too attenuated, a very marked "break" occurs, because of the vast differences in density of the adjoining media.* As far, then, as different media in the chest affect sound intensity by their combination, they may be roughly reduced to two: a medium of air and a medium free from air.

To summarize what has been said about the diminution of sound in the chest, we are dealing chiefly with two factors—diffusion and reflection—the former occurring fairly constantly, the latter for the most part requiring the presence of air and taking place mainly at the junction of air with either solid (tissue, gel), or fluid. Or, to put it in another way, there is very little diminution of sound in the absence of air, except that due to diffusion, provided the bronchi are patulous and not reduced in size.

In speaking of the combination of tissue and air as offering an obstruction to the passage of sounds, the degree of obstruction will depend on the thickness of the tissue adjacent to the air. Thus, much more sound is lost when the vibrations are passing from air to chest wall, than from air in one of the vesicles in the lung to the delicate enveloping membranous wall. The fact that sounds pass though the normal lung with as little loss as they do, is largely because each delicate membrane individually offers relatively very little obstruction. *More obstruction occurs when these membranes are tense than when relaxed, the greater tenseness of the membranes in the former case effecting a greater degree of reflection of the vibrations.* The result is the well known clinical fact, that the normal lung in the normally distended state impedes the transmission of sound more than when in a state of collapse. *A great difference in transmissability can be shown to exist when the sounds traverse equal distances in distended and relaxed lungs.*

While the solid tissues of the lung (normal and diseased), and of the chest wall in the living body contain a large proportion of fluid in the form of lymph and blood, this mixture of two different media, fluid and tissue, does not materially reflect or obstruct the passage of sounds, for the reason given, that the densities of tissue (normal and pathological) and fluid are relatively alike.

The purely solid portions of the tissues we are considering are made up chiefly of soft cellular substance, which, to a large extent at least, is physically allied to gelatin, and belongs to the group of gels, substances which it is well to bear in mind are very different physically from such solid materials as wood and metal. These gels, though potentially capable of yielding a large proportion of fluid, do not actually contain it, just as gelatin, though capable of yielding much water, does not do so as long as it is in the state of gelatin.

ABSORPTION, RESONANCE AND INTERFERENCE OF SOUNDS. Besides the main factors of diffusion and reflection of sounds there are other factors affecting the intensity of sounds within the chest, but their activities, what little we know about them, appear to be much less influential. On the quality of sounds, however, at least in the case of resonance, their effect may be very marked. By *absorption* is meant the annihilation of sound energy as such, and its conversion into other forms of energy, such as heat, which may not be appreciated by the ear. It is a factor concerned in the diminution of sound intensity quite distinct from diffusion, though at times confused with it. Absorption by different media apparently plays a very small part in the reduction of sound intensity in the chest. *Resonance* acts particularly by affecting sounds of a certain pitch, and as the chest sounds familiar to us are compounded of many different kinds of individual sounds, resonance usually exerts its influence only on a limited number of these individual sounds. Certain elements in the original sound may be diminished through *interference*, that is, the superimposition of sound waves upon each other.

The Size of the Bronchi as a Factor in Determining the Intensity of the Vocal Resonance. When a bronchus, or a group of bronchi, such as we find in the bronchial tree of one lung, become reduced in size, the total amount of sound energy coming from the upper respiratory tract and being admitted to these bronchi, is less than when the bronchi possessed their original larger capacity. How much of an effect this decrease in the bronchial capacity may exert upon the vocal resonance requires further investigation. This factor would have a particularly favorable opportunity to operate when the whole lung is much reduced in size, as may occur in pleural effusion or pneumothorax. Braune and Stahel[1] have shown that the diameter of a bronchus usually depends in part upon the amount of air in the lung, and it seems quite possible that in chronic affections of the lung and pleura the bronchial capacity might become sufficiently reduced to appreciably affect the vocal resonance. The explanation for the greater vocal resonance often encountered normally over the right lung, may rest partly on the fact that the right lung is larger than the left, which necessitates a larger total intrabronchial capacity on the right side, which permits the entrance of a larger amount of sound energy into this side. (The ratio of the diameters of the right and left main bronchi in the normal individual, according to Braune and Stahel, is about 4 to 3, approximately the same ratio that they obtained for the respective weights of the lungs on the corresponding sides.)

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[1] Ueber des Verhältniss der Lungen, als zu ventilirender Lufträume, zu den Bronchien, als luftzuleitende Röhren, Arch. f. Anat. u. Physiol., 1886, Anat. Abtheilung, S. 5.

THE ORIGIN OF THE SOUNDS INVOLVED. It is to be recalled that the spoken voice sounds, bronchial breathing, and whispering pectoriloquy arise in the upper respiratory tract, so that the source of these sounds is identical, however variable may be the conditions in the lungs, and different intensities in different parts of the external chest surface must be due to factors operating distal to the source of these sounds. On the other hand, variations in intensity of the vesicular breath sounds, at least of the inspiratory ones, sounds which arise within the lungs, may be due in whole or in part to differences in intensity at the site of production of these sounds. Thus, in pleural effusion, even if slight, the vesicular murmur may be weakened because there is not proper expansion of the vesicular structure to produce sounds of normal intensity.

THE RELATIVE IMPORTANCE OF DIFFERENT PORTIONS OF THE RESPIRATORY PASSAGES BELOW THE LARYNX AS DIRECT SOURCES OF THE VOCAL RESONANCE. While the trachea and bronchi resemble speaking tubes to the extent that they transmit within their interior sounds coming from the upper part of the respiratory tract toward peripheral portions of the lungs, the walls of the tracheobronchial tree are, all along the route, taking up a certain amount of the sound present within these air passages. Some of the vibrations entering these walls are never perceived by the auscultator, and, in the case of the normal lung, the vocal resonance over most of the chest is due entirely or largely to vibrations escaping from the smaller bronchi. Under these conditions the trachea and larger bronchi serve only as carriers of sound from the upper respiratory tract to the smaller bronchi. The normal right apex, according to Fetterolf,[2] receives some audible vibrations that have come directly from the trachea without having had to pass through the bronchi. In pneumonia it has been shown by Mason[3] that the voice and breath sounds become much altered when a peripherally situated consolidation, previously separated from the large bronchi by an area of normal lung tissue, by extension centrally, finally comes in direct contact with these large bronchi. The large bronchi furnish some of the sound when the lung assumes the fetal type following the disappearance of air from within the alveoli. The trachea itself may be a direct source of sound when the upper part of the lung is solid, or when the upper portion of the pleural cavity is filled with air or fluid, the pulmonary apex being free from adhesions and retracted toward the root of the lung.

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[2] The Anatomic Explanation of the Greater Amount of Vocal Fremitus and Vocal Resonance Normally Found at the Apex of the right Lung, Archives of Internal Medicine, February, 1909.

[3] American Journal of Diseases of Children, March, 1916.

While fully recognizing the limitations of the explanations offered for various auscultatory findings obtained over the lungs, we believe that the application of the simple principles just presented to certain clinical conditions can frequently be determined to a considerable extent. Six of these conditions will receive brief attention: solidified lung, normal lung in a state of normal distention, collapsed but otherwise normal lung, pleural effusion with diminished vocal resonance, pleural effusion with increased vocal resonance, and pneumothorax.

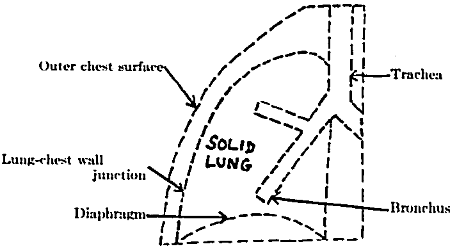

FIG. 1.—Solid lung. No special reflection, the entire distance between bronchial wall and outer chest surface. Diffusion an important but not a special factor. More sound retained within the bronchi, at least, the small ones, than normally. Vocal resonance increased. Bronchi in diagram represent small as well as large bronchi.

Solid Lung. This is the simplest clinical condition we have to study. In tracing the sounds from within the bronchi through the solid lung to the periphery of the chest we find the factor of diffusion of sound everywhere operative. The activities of this factor are very much limited within the bronchi because the bronchial walls to a great extent reflect the vibrations back into the interior of the bronchi, this reflective action in the smaller bronchi being even more powerful when the lung is solid than when it is normal. Once the vibrations have entered the bronchial walls, however, their further course is pursued entirely through tissue with its associated fluids, the solid lung and chest wall forming practically a single good conducting medium which offers very little obstruction to the passage of the vibrations, so that *nearly all the sound intensity that is lost en route from bronchus to chest periphery is due to diffusion.* In no other common clinical condition do we find so little tendency to the reduction of sounds from reflection as when dealing with a solid lung. And even the degree of diffusion is not greater than that ordinarily encountered. The voice sounds are, therefore, relatively loud, or, as we usually express it, are increased in intensity over a solid lung. In addition the weaker sounds produced in the upper respiratory tract, bronchial breathing and whispering pectoriloquy, become audible at the chest surface, while the normal breath sounds disappear because they are not produced.

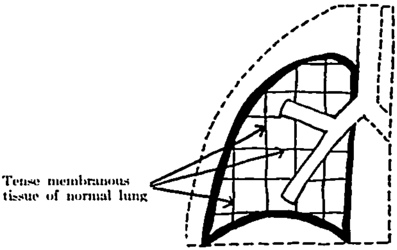

FIG. 2.—Normal lung. Special reflection (indicated by continuous lines), first, at junction of the air in the lung with the chest wall; second, at junction of tense membranous tissues of lung with air in the vesicles, and, possibly to some extent; third, at junction of bronchi with air in adjacent vesicles. Diffusion much as in the case of solid lung. Vocal resonance normal. Bronchi in diagram represent small as well as large bronchi.

The Normal Lung Normally Distended. The conditions present in a chest containing normal lung in its normal state of tension offer a marked contrast to those occurring when the lung becomes solidified. Such a lung, unlike the solid lung, does not exhibit a single good conducting medium free from air between bronchial wall and chest wall. Instead, we find air associated in a very peculiar way with the delicate membranous tissues of the pulmonary parenchyma, these membranes at the same time being under a considerable degree of tension. Sound energy is lost, (1) as the vibrations pass from the bronchial walls to the air in the nearest adjoining vesicles; (2) as the vibrations pass between the air in the vesicles and the tense membranous walls enclosing them, and (3) as the vibrations pass from the air in the vesicles immediately underlying the chest wall to the tissues between these vesicles and the chest wall, which practically means to the chest wall. *Each such loss of energy is the result of reflection* acting on the vibrations as they pass toward the exterior of the chest. It is not surprising, therefore, with all these places unfavorable to the transmission of sound, that the normal vocal resonance is so much less intense than when the lung is solid. In fact, it is more difficult to explain why the voice sounds are not actually weaker than they are. Two conditions may be mentioned which are relatively favorable to sound transmission when the lung is normal: (1) Sound can more easily escape from within the smaller bronchi to the thin walls of these smaller bronchial tubes of the normal lung than when these tubes become encased in solid tissue. (2) There is some sound conduction even in the thin membranous tissue of the normal lung. The absence of bronchial breathing and whispering pectoriloquy over normal lung is due to the same conditions that make the vocal resonance less than that over solid lung.

Relaxed but Otherwise Normal Lung. Clinically this condition occurs above a pleural effusion and is apparently responsible for the increased vocal resonance frequently found above an effusion. A good deal of the confusion and error that has resulted in the attempt to explain different degrees of intensity in the sounds heard over the chest has been due to a failure to recognize *the different effects on sound transmission occasioned by the normally distended lung and the collapsed lung*, or, as it is frequently termed, the compressed lung. Bullar (1884) was the first one to describe the marked differences in conductivity of these two kinds of lungs, though he offered no explanation for this variation. *The relaxed lung allows vibrations to pass through it with less obstruction or reflection than the distended lung,* for the reason given in speaking of the normally tense lung, that is, *because the relaxed membranes of the collapsed lung are not under tension,* and therefore reflect the vibrations backward to a much less extent than when the same membranes are tense. This principle is well known, and can be readily illustrated by passing sounds into a tube and listening at the far end, when it will be noticed that if a thin piece of rubber membrane (or body tissue) is placed across the lumen of the tube at some point, much less sound gets through to the far end of the tube when this membrane is held taut than when it is completely relaxed. The relaxed lung lets more sound through it than the distended lung even when the area traversed in the two cases is identical. In all respects, *except the degree of tension of the finer tissues,* the relaxed and tense lungs are very much alike as far as sound transmissibility is concerned, when equal distances in the two kinds of lungs are compared.

Pleural Effusion with Diminished or Absent Vocal Resonance. The explanations for this phenomenon when not entirely erroneous have been either obscure or incomplete. The abnormal conditions encountered in a simple case are the presence of fluid within the pleural cavity, and a more or less relaxed lung. The clinical fact above referred to, that the vocal resonance above a pleural effusion is frequently increased, indicates that the sounds escape from the relaxed lung to adjacent tissue with more intensity than in the normal case. The evidence indicates that the sounds pass between a relaxed lung and fluid with about an equal intensity to that found in the passage of sounds between relaxed lung and tissue. Therefore, in those cases presenting an increase in vocal resonance above an effusion we conclude that the vibrations entering the fluid are louder than those entering the chest wall of an individual with the lung in a normal state. *Any loss of sound, then, (except as noted below), encountered over a pleural effusion, must take place after the vibrations have entered the fluid.* We have already seen that tissue alone, and fluid alone, and even the combination of the two, offer very little impediment to the passage of sounds. *The loss of sound encountered over an effusion may be traced largely to diffusion*, the result of the greater distance occurring between the lung and the chest wall overlying the fluid as compared to the close approximation of the lung to the chest wall under normal conditions when no fluid separates them. The sounds become weak over an effusion for the same reason that they do over the liver, because of diffusion and not because of reflection. When the lung is much reduced in size the diminished vocal resonance may be partly due to the decrease in size and capacity of the bronchi, which allows less sound to enter the lung than normally. When effusion is present the vesicular murmur may be diminished or absent partly because the vesicles are not expanding properly to produce sounds of normal intensity.

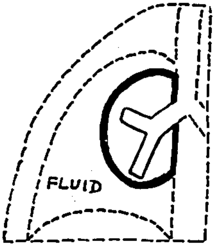

FIG. 3.—Pleural effusion. Lung air-bearing, but membranous tissue relaxed. Special reflection (indicated by heavy continuous line) at lung-fluid junction. Reflection at junction of bronchi with adjacent intravesicular air (indicated by light continuous lines) of questionable importance. Special diffusion between lung and chest wall. Vocal resonance decreased. Bronchi in diagram represent small as well as large bronchi.

Pleural Effusion with Increased Vocal Resonance. The explanations offered to account for an increase in the vocal resonance sometimes met with over a pleural effusion are few and for the most part unsatisfactory. *In some cases this increased vocal resonance is apparently due to the fact that the fluid adjoins a lung which is solid.* The lung may be solid simply from absence of air in the vesicles, as when it assumes the fetal type after prolonged collapse, or it may be solid as a result of inflammatory or other products in the vesicles. It is important that no air should remain in the pulmonary parenchyma. *When a solid lung is separated from the chest wall by fluid there is very little reflection or other form of obstruction to the passage of vibrations on their way to the periphery of the chest, after they have entered the bronchial walls. Neither the tissues (lung and chest wall), nor the fluid, either individually or in combination, offer any serious impediment to the passage of sounds.* If, then, diffusion is limited, the conditions are very much as in the case of the solid lung unassociated with fluid. And whatever the degree of diffusion, due to separation of the lung from chest wall and other factors, the conditions are very much the same as though tissue replaced fluid in the pleural cavity. The explanation is entirely satisfactory where the solid lung is separated only a short distance from the chest wall by the fluid, but it is well known that very large effusions on either side of the chest separating the lung a great distance from the chest wall, and thereby greatly increasing the diffusion between lung and chest wall, may furnish striking examples of cases with increased vocal resonance. In such cases it seems likely that some vibrations are received at the periphery of the chest *coming straight across through the fluid from the trachea.* The physical conditions are certainly favorable for this,

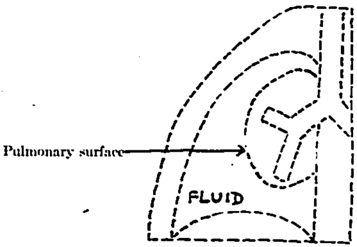

Fig. 4.—Pleural effusion. Lung solid. No special reflection, the entire distance between bronchial wall and outer chest surface. Compare solid lung. Special diffusion may exist between lung and chest wall. Vocal resonance may be increased. Bronchi in diagram represent small as well as large bronchi.

that is, the fluid may be present in the upper part of the pleural cavity, so that in the course of the vibrations from the trachea to the chest wall no air intervenes, but only tissue and fluid. Very loud sounds may be obtained over the chest of a fetal calf after fluid has been introduced between the chest wall and the solid fetal lung, these sounds becoming much weaker, however, after inflation of the lung. (The sound in this case is introduced into the trachea.)

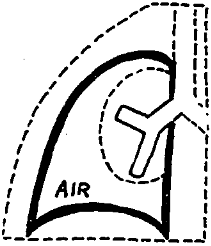

FIG. 5.—Pneumothorax. Lung air-bearing. Special reflection (indicated by heavy line) at air chest-wall junction. Reflection at outer bronchial surface as in Fig. 3. No special diffusion; diffusion being limited by the heavy walls enclosing the air in the pneumothorax cavity. Vocal resonance distinguished. Bronchi in diagram represent small as well as large bronchi.

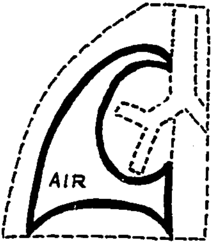

FIG. 6.—Pneumothorax. Lung solid. Same as Fig. 5, except that there is special reflection (indicated by heavy continuous line) between solid lung and surrounding air, while there is no reflection between outer surface of bronchi and surrounding solid tissue. Vocal resonance diminished. Bronchi in diagram represent small as well as large bronchi.

Pneumothorax. In the case of pneumothorax the distinctive location of sound loss is at *the junction of the air in the pneumothorax cavity with the chest wall,* where reflection of the vibrations acts very powerfully as a result of the enormous differences in density of air and tissue. If the collapsed lung is air-bearing the opportunities are more favorable for the transmission of sounds to the periphery of the chest than if the lung is solid, because more sound can enter and leave a collapsed air-bearing lung under these conditions than when the lung is solid, while at the same time the air-bearing lung itself is a very good transmitter of sound. If no adhesions exist at the apex sound may be obtained coming directly from the trachea to the chest periphery by way of the pneumothorax cavity. *Diffusion exerts little effect between the lung and the chest wall*, the sound intensity being fairly equal throughout the pneumothorax chamber, because the air is enclosed within solid walls. The diminished vocal resonance is probably partly due to the small size of the collapsed lung and the consequent diminished calibre of its bronchi, resulting in the admission of a reduced amount of sound into the affected lung. When a patulous fistula exists between the pneumothorax cavity and a bronchus, the conditions are favorable for abnormally loud vocal resonance.[4]

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[4] Some of the subjects briefly presented in this paper have been treated more fully in the Tenth Report of the Henry Phipps Institute for the Study, Treatment, and Prevention of Tuberculosis, in an article by Montgomery and Eckhardt, entitled "Pulmonary Acoustic Phenomena."